Results: We investigated 64 institutionalized children aged 2 to 12 years. Parasitic infections were identified in 44 cases (68.7%). *Hymenolepis nana* (31.2%), *Giardia lamblia* (28.1%), *Blastocytis hominis* (7.8%), *Entamoeba coli* (4.7%), *Ascaris lumbricoides* (4.7%), and *Trichuris trichiura* (3.1%) were diagnosed. We have determined associations of two (45.4%) and more than two parasites (18.2%) among the infected children; of the 20 with hymenolepiasis, 11 (55%) had multiple parasitic infections. Clinical and laboratory examinations were conducted to investigate the presence of symptoms and eosinophilia in patients with parasitic infections. Diarrhoea (38.6%), weight loss (47.7%), abdominal pain (25%), nervous disorders (34.1%), cutaneous manifestations (22.7%), fever (9.1%) and respiratory infections (59.1%) were reported in the infected children. Eosinophilia was noted in 27 (61%) of the 44 patients with parasitosis. High eosinophil values were mostly observed among those diagnosed with helminth infections.

Conclusion: Hymenolepis nana infection was reported with an increased frequency among Romanian institutionalized children. The majority of the patients with hymenolepiasis were diagnosed with multiple parasitic infections.

doi:10.1016/j.ijid.2010.02.2140

58.017

Computational characterization of molecular mechanism of surface receptor binding with alpha-enolase of *Trichomonas vaginalis*

S. Dwivedi[1,*], S. Mishra[2], N. Husain[3], N. Malla[4]

[1] *CSM Medical University, Lucknow, India*
[2] *College of Engineering & Technlology, Moradabad, UP, India*
[3] *CSM Medical University, Lucknow, UP, India*
[4] *Postgraduate Institute of Medical Education and Research, 160012, Chandigarh, India*

Background: Trichomonas vaginalis is a parasitic protozoan causing the most common human sexually transmitted disease (STD), trichomoniasis. The exact mechanism of its pathogenesis is still obscure. Alpha-enolase plays a pivotal role in the host-pathogen interaction, and as a surface receptor of several protists mediating plasminogen binding. In view of identifying plasminogen binding sites of *T. vaginalis* alpha-enolase, homology modeling and docking studies were conducted to obtain modeled structures of the *T. vaginalis* alpha-enolase-plasminogen complex.

Methods: The protein sequence of *T. vaginalis* alpha-enolase used in this study was kindly provided by Dr. John F. Alderete (University of Texas, USA). Modeling templates were searched by using BLAST, followed by multiple sequence alignment. The atomic coordinates of *Escherichia coli* enolase was retrieved from Protein Data Bank. Molecular structures of *T. vaginalis* alpha-enolase were modeled by using restraint-based modeling, followed by energy minimization using MODELLER program. The quality and stereochemistry of the models were evaluated by program PROCHECK. After addition of Mg2+, the selected model further refined by energy minimization employing NAMD program. The VMD program was used to superimpose structure of *T. Vaginalis* alpha-enolase model with crystal structures of enolases from *E. coli* and *S. pneumoniae*. *T. vaginalis* alpha-enolase model was docked to human plasminogen for protein-protein interaction using Hex 5.1. Mark Gerstein's calc-surface program was used to calculate the solvent accessibility at the interface of *T. vaginalis* alphaenolase and human plasminogen before and after docking.

Results: Molecular docking revealed hydrogen bonding of eLys70-pgTyr50, eAsn165- pgThr66, eAla168-pgGlu21, eAsp17-pgLys70, and eAsn213-pgPro68/pgAsn69. Substantial decreases in accessible surface area (ASA) were observed and in concurrence with hydrogen bond pattern.

Conclusion: These findings provide new insights for interaction at the protein-protein interface. Our theoretical prediction is consistent with preexisting biochemical data. The predicted interaction complex can be of great assistance in understanding structural insights, probably being necessary to an interaction between pathogen and host-component. The ability of *T. vaginalis* alpha-enolase to bind plasminogen may be indicative of being a key player in invasion of this pathogen to host. Conclusively, this work theoretically establishes the *T. vaginalis* alpha-enolase as a novel surface-linked virulence factor.

doi:10.1016/j.ijid.2010.02.2141

58.018

Prevalence of *Dientamoeba fragilis* among asymptomatic individuals from North Central Venezuela

S. Jimenez[1], J. Cortez[1], M. Diaz[1], C. Duran[2], G. Hidalgo[3], W. Aguilera[4], S. Nakal[4], C. Albano[5], R.N. Incani[1], A. Rodriguez-Morales[6,*]

[1] *Department of Parasitology, Faculty of Health Sciences, Universidad de Carabobo, Valencia, Venezuela*
[2] *Coordination of Coproparasitology, Direction of Biological Sciences, Foundation Center for Studies on Growth and Development of the Venezuelan Population (FUNDACREDESA), Ministerio del Poder Popular para las Comunas y Protección Social, Caracas, Venezuela*
[3] *Direction of Biological Sciences, Foundation Center for Studies on Growth and Development of the Venezuelan Population (FUNDACREDESA), Ministerio del Poder Popular para las Comunas y Protección Social, Caracas, Venezuela*
[4] *Coordination of Statistics, Direction of Population Studies, Foundation Center for Studies on Growth and Development of the Venezuelan Population (FUNDACREDESA), Ministerio del Poder Popular para las Comunas y Protección Social, Caracas, Venezuela*
[5] *General Direction of Research, Foundation Center for Studies on Growth and Development of the Venezuelan Population (FUNDACREDESA), Ministerio del Poder Popular para las Comunas y Protección Social, Caracas, Venezuela*
[6] *Direction of Population Studies, Foundation Center for Studies on Growth and Development of the Venezuelan Population (FUNDACREDESA), Ministerio del Poder Popular para las Comunas y Protección Social, Caracas, Venezuela*

Background: Dientamoeba fragilis is a protozoan parasite, with the presence of only trophozoites in its life cycle, with worldwide distribution, and that may be considered responsible for enteric disease in humans. Although described in 1918 by Dobell, many clinical and pathological